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EXPERIMENTAL RESEARCH INTO HIGH BAROMETRIC OXYGEN PREVENTION OF GUINEA PIG HEARING LOSS

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ABSTRACT

Two groups of guinea pigs were exposed to an acoustical field of 125 dBSPL one kilohertz for three hours. One of these groups breathed high barometric oxygen at two atmospheres for three times prior to exposure and on 21 continuous days following exposure. They breathed this pure oxygen for one half hour each time. The other group was control and was not given pure oxygen. The indexes used to judge the results wee induced potential hearing threshold of the hearing regions of the cerebral cortex and cochlea pathology. After exposure to sound, the average hearing loss of the high barometric oxygen group was 35 to 50 dB. That of the control group was 60 to 70 dB ($P < 0.01$). The average total length of cochlear damage and the length of serious damage of the high barometric oxygen group were 80 microns and 34 microns while that of the control group was 267 and 210 microns respectively ($P < 0.01$). The results indicate that high barometric oxygen acts to prevent and cure hearing loss.

KEY WORDS: High barometric oxygen, tearing loss, guinea pig, cerebral cortex hearing area induced potential, and cochlea pathology.

CONDITIONS AND METHODS

I. EXPERIMENT ANIMALS AND DIVISION OF GROUPS

We selected 30 guinea pigs weighing 280±40 grams, with normal ear drums and with high concha sensitivity. The guinea pigs were separated into three groups by weight and sex. These three groups were the high barometric oxygen prevention and treatment group, the sound stimulation control group, and the normal control group. The experiment conditions and the division of the animals are shown in table one.

TABLE ONE: CONDITIONS AND MEASUREMENT INDEXES OF THE THREE GROUPS

表 1 三组动物的实验条件与测定指标(只)

	豚鼠只数	实验条件		检查指标	
		声暴露	高压氧	耳病理	听力电位
正常对照组	10	--	--	10	4
声刺激对照组	10	10	--	8	6
高压氧防治组	10	10	10	8	6

1. Number of guinea pigs. 2. Experiment conditions. 3. exposure to sound. 4. High barometric oxygen. 5. Inspection indexes. 6. Ear pathology. 7. Hearing potential. 8. Normal control group. 9. Sound stimulation control group. 10. High barometric oxygen prevention and treatment group.

II. DISTRIBUTION OF ANIMALS IN THE ACOUSTICAL FIELD

A high intensity pure sound of one kilohertz at 125 dB SPL was produced by a Denmark model 1027 sine wave generator connected to a domestically produced Meiduo Brand model 250 amplifier and a domestically produced model HDZ-200-241 C loud speaker. The intensity and frequency of the sound was monitored and measured by a Denmark model 4131 capacitance microphone and a Denmark model 2112 spectrograph.

The animals placed in the acoustic field were all placed individual wire cages and wire was tied around their necks to keep them from shaking their heads. The animals were placed with their heads facing the sound source, and the monitoring and measurement microphones were placed beside the testing animals.

The animals in the sound stimulation control group and the high barometric oxygen prevention and treatment group were exposed to the sound for three hours.

III. CONDITIONS FOR AND NUMBER OF TIMES OXYGEN WAS GIVEN

The conditions for the supplying of oxygen was such that it did not damage the ear drum or cause hemorrhaging of the mucous membranes of the tympanic cavity. After some trials, we determined that they would be given high barometric oxygen at two atmospheres of pressure (actually one kilogram per square centimeter). During actual operations, the pressure was evenly increased to one kilogram per square centimeter over 30 minutes, kept at this level for 30 more minutes, and then pressure was reduced to normal over ten minutes. Prior to pressurization, the inside air of the equipment was rinsed out with oxygen so the oxygen concentration inside the equipment was the same as that in the oxygen tanks (99.2 to 99.5 percent). The carbon dioxide concentration was four

to five percent.

The animals breathed oxygen once prior to noise exposure and once a day for 21 days following exposure. After another 11 days, the animals were killed and their inner ears extracted.

IV. OBSERVATION INDEXES

1. Induced potential at cerebral cortex hearing areas: (1). We used a British Medelec/Amplaid MKIII ERA complex instrument to measure the induced potential of the guinea pig cerebral cortex. Short bursts of pure sound and short acoustic signals of 0.5, 1.0, 2.0, 4.0 and 8.0 kilohertz were transmitted by the excitor. The duration of the bursts of pure sound was 100 ms, and the rise and fall each took 10 ms. The duration of the short acoustical signals was 100 microseconds and the frequency was 0.8 to 5.0 kilohertz. The two acoustical signals were then transmitted by a model TDH 39 hearing aid at a distance of 0.5 centimeters from the ear canal. (2). Number of times induction electrodes were implanted and tested: The electrode implantation was primarily done using the technique of the Shanghai Institute of Biology Hearing Team[4]. The induction electrodes were implanted at the hearing areas on either side of the animals' heads. The reference electrode was implanted at an incision in the scalp. The ground was placed inside the mouth of the animal. The experiment was conducted inside a soundproof room with electronic screening. During the hearing tests, we primarily observed the value of the induced potential at the hearing areas of the cerebral cortex, and the potential near the threshold, was superimposed 32 and 64 times using a model DAV 62 equalizer. The evaluation of the hearing threshold level was done by four people working together. The cerebral cortex hearing area induced potential tests were conducted prior to sound exposure and on the first, third, fifth, eleventh, 25th and 32nd day; following exposure. The normal group was tested on the 12th and 28th day. The electrodes were put in place just prior to the testing.

The inner ear samples were prepared according to the Schuknecht method, wrapped in collodion, and consecutive slide samples were taken along the modiolus. The slide sample thickness was 20 microns, died with HE, and checked with an optical microscope. We recorded the locations and extent of damage to the inner ear of guinea pigs and calculated the length of the damage to the inner ear[5].

RESULTS

I. CEREBRAL CORTEX HEARING AREA INDUCED HEARING THRESHOLD POTENTIAL

1. Cerebral cortex hearing area induced hearing threshold potential prior to exposure to sound: The mean hearing threshold for the normal group, the stimulated control group and the high barometric oxygen group were basically very close. The individual frequency hearing threshold averaged differed from five to seven decibels ($P > 0.05$). The hearing threshold of all the animals in the three groups fell within the normal

range. The normal control group had basically the same hearing threshold on the first and the 28th day following the experiment, with variations of from two to four decibels at the various testing points.

2. Changes in cerebral cortex hearing area induced hearing threshold potential following exposure to sound: The average hearing threshold at various frequencies in the short pure sound test and the short sound blast hearing threshold of the acoustically stimulated control group was as much as 80 to 107 decibels. Hearing loss was 60 to 70 decibels. There was also a rise in the hearing threshold of the high barometric oxygen group, where hearing loss was 30 to 50 decibels, but there was a very marked difference in the amount of hearing loss between the two groups. Between three to five days after exposure to sound, the hearing of the two groups recovered fairly quickly. Later, recovery slowed, and after the eleventh day, there was no further recovery (see illustration one).

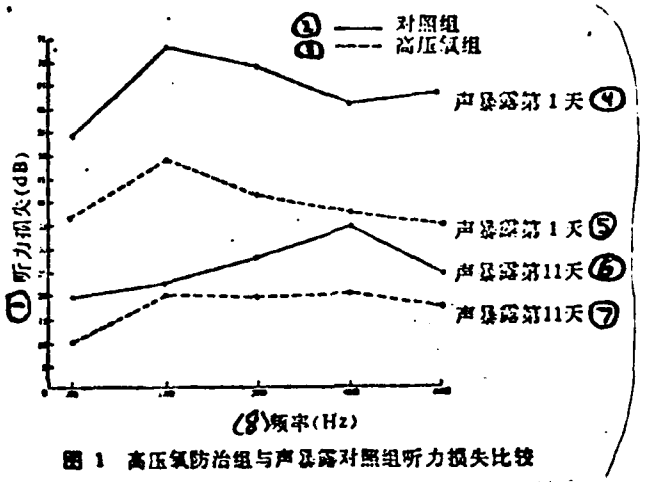

图 1 高压氧防治组与声暴露对照组听力损失比较

ILLUSTRATION ONE: COMPARISON OF HEARING LOSS OF HIGH BAROMETRIC OXYGEN PREVENTION AND CURE GROUP WITH NOISE EXPOSURE CONTROL GROUP

1. Hearing loss (in decibels). 2. Control group. 3. High barometric oxygen group. 4. First day after exposure. 5. First day after exposure. 6. Eleven days after exposure. 7. Eleven days after exposure. 8. Hertz.

The difference in hearing loss between the sound stimulation control group and the high barometric oxygen group was statistically processed with the following results:

Table two shows that the hearing threshold of the two groups was about the same two days prior to acoustic stimulation. There was a statistically significant difference in the hearing thresholds of the two groups on the first, third, fifth and eleventh day following the stimulation. This was due to the hearing loss prevention and cure effects of high barometric oxygen. On the 25th and 32nd day following stimulation, there was no clear significant difference at the various testing points. This is related to the effects on hearing of the occurrence of inner ear infections in the animals of the high barometric oxygen group. This can be seen in the average hearing charts of the five animals (because one animal died) which had inner ear compared with the

charts of the four animals which did not have inner ear infections (see illustration two). The prevention and treatment effects of high barometric oxygen were still maintained on the 32nd day following acoustic stimulation.

表 2 高压氧防治组与声刺激对照组听力差别显著性测验(t值)

测试频率 (Hz)	声刺激前	声刺激后					
		1[A]	3	5	11	25	32
500	0.835	2.699**	2.556**	2.653**	2.545*	0.094	0.500
1000	0.000	4.611**	2.486*	4.524**	0.893	1.017	0.906
2000	1.710	4.562**	3.609**	3.226**	1.322	0.084	0.856
4000	1.771	4.260**	4.311**	4.034**	2.960**	0.713	1.099
8000	1.465	4.270**	3.766**	3.406**	1.023	0.545	0.812
短 声	1.970	4.084**	5.474**	5.519**	2.964**	0.777	0.233

** $P<0.01$ * $P<0.05$ A 横栏为天数

TABLE TWO: TEST FOR AMOUNT OF DIFFERENCE IN HEARING BETWEEN THE HIGH BAROMETRIC OXYGEN GROUP AND ACOUSTIC STIMULATION CONTROL GROUP

1. Testing frequency (in hertz). 2. Prior to acoustic stimulation. 3. Following acoustic stimulation.

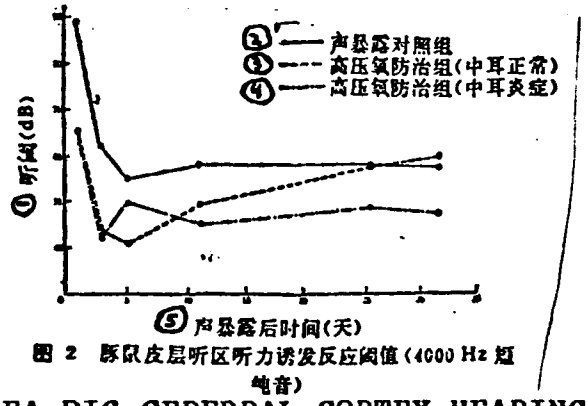

图 2 豚鼠皮层听区听力诱发反应阈值(4000 Hz 短纯音)

ILLUSTRATION TWO: GUINEA PIG CEREBRAL CORTEX HEARING AREA INDUCED HEARING REACTION THRESHOLD (SHORT BURST OF 4,000 HERTZ PURE SOUND)

1. Hearing threshold (in decibels). 2. Sound exposure control group. 3. High barometric oxygen prevention and treatment group (with normal inner ears). 4. High barometric oxygen prevention and treatment group (with middle ear infections). 5. (Days) following exposure to noise.

We should point out that the middle ear infections are not side effects of high barometric oxygen treatment. Practice has demonstrated that if the animals are protected against catching colds and their eustachian tubes are kept clear, the middle ear infections can be avoided. We also conducted high barometric oxygen treatments on 36 guinea pigs, and because we were careful to follow the steps described above, none of these guinea pigs had middle ear infections.

II. PATHOLOGICAL CHANGES IN THE MIDDLE EAR

1. Differentiating the degree of damage in the ear canal. In light of the experience of our laboratory anc combining this with published reports, differentiation was done according to the function of the internal and external capillary cells and their tolerance to noise[6,7], dividing the hearing loss into three levels.

Serious hearing loss: On the basis of changes in the external capillary cells, damage and loss of internal capillary cells occurred. Furthermore, damage, reduction or loss frequently occurred to the spiral organ and the spiral organ nerve of the inner ear (see illustration three. Translator's note: not available in article).

Intermediate hearing loss: Total damage to or loss of ear canal spiral organ external capillary cells or their nuclei, may be accompanied by damage or loss of supporting cells (see illustration four. Translator's note: not available in article).

Slight hearing loss: One or two of the external capillary cells of the spiral organ of the ear canal have been damaged ar show loss of nucleus loss or high concentration of nucleus or serious nucleus shift in a portion of the external capillary cells (see illustration five. Translator's note: not available in article).

The hearing loss levels could be further delineated, but for the sake of simplicity, we believe that three levels are enough to satisfy the requirements of our experiment.

2. Number of guinea pig ears damaged: We examined 16 ears of guinea pigs in the acoustic stimulation control group, and there was damage to eight of these. In the high barometric oxygen group we also examined 16 ears, but only three ears showed cochlear damage. The number of ears showing damage in the second group is clearly fewer than those in the first group.

3. Length and degree of guinea pig cochlear damage (see illustration six): The average length of accumulated cochlear damage in the animals in the acoustic stimulation control group was 267 microns. In the high barometric oxygen group, the average length of accumulated cochlear damage was 84 microns. There was not a great deal of difference between the damage length between the animals in the two groups with slight or intermediate damage. Of the animals with serious damage, the average damage to the animals in the acoustic stimulation control group was 201 microns and to the animals in the high barometric oxygen group the average was 34 microns. Through statistical analysis (t test), there is an extremely obvious significance ($P < 0.01$) between the acoustic stimulation control group and high barometric oxygen group in total length of cochlear damage and degree of length of serious damage. The breathing of high barometric oxygen also acts to provide excellent prevention in

cochlea morphology aspects.

DISCUSSION

There have already been experiments which indicate that there is rapid metabolism and high oxygen consumption in the cochlea tissue[8], and that this tissue is sensitive to oxygen deprivation[7]. Exposure to intense sound levels can cause the blood vessels of ears in animals to constrict and narrowing of capillaries[1,8]. It can also cause a drop in the oxygen content of the lymph in the inner and outer ear[2,10]. Biopsy analysis shows that there is an increase in the lactic acid content in the inner ear[11], indicating that during exposure to noise, cochlea metabolism tends toward anaerobic metabolism, and the cochlea conversion of sound into nerve impulses is an energy consumption process. As everyone knows, anaerobic yeast reduction yields much less energy than aerobic metabolism. It may be inferred that intense noise stimulation causes an excess oxygen debt in the cochlea spiral organ, and cause in the cochlea capillary cells, like other oxygen deprivation sensitive cells, possible varying degrees of hindrance toward their metabolism, or even acid poisoning, lysosome cracking and finally lead to serious damage to the cells or death of the cells. There are also experiments which have demonstrated[12] that breathing oxygen can increase the oxygen pressure in the cochlea lymph fluid. By mixing a certain ratio of carbon dioxide in with the oxygen, it is also possible to cause the expansion of the microscopic blood vessels, which helps oxygen to be dispersed to the cochlea tissues. We can believe that timely inhalation of high barometric oxygen prior to or following exposure to intense noise should be beneficial to preventing and curing cochlea noise damage, because advance inhalation of oxygen can increase the oxygen storage of the cochlea tissue and oxygen inhalation after the fact can supplement localized oxygen insufficiency.

Red blood cells (hemoglobin) transport 96.4 percent of the oxygen in the blood, and serum dissolved oxygen only 3.6 percent. Breathing pure oxygen at normal pressures will elevate the serum supply of oxygen to the tissues to as much as 33.9 percent. This is because the serum dissolved oxygen follows the laws of physics. If pure oxygen is inhaled at a pressure of two absolute atmospheres, the serum dissolved oxygen can reach 73.2 percent, and at pressures of three absolute atmospheres, it can reach 94.6 percent, thus completely replacing the oxygen transport role of the blood cells. Under conditions of high barometric oxygen, it is also possible to increase the capillary blood oxygen dispersion radius from 30 microns to 100 or 1000 microns, thus making up for the deleterious effects of intense noise and those of pure oxygen itself of causing constriction of microscopic blood vessels[13]. Joglekar[3] points out that inhaling pure oxygen or pure oxygen mixed with five percent carbon dioxide can reduce temporary hearing thresholds caused by noise exposure (one kilohertz at 100 decibels SPL). Our experiment has shown that breathing high barometric oxygen can prevent or reduce permanent hearing loss caused by exposure to noise (one kilohertz at 125 decibels SPL). We can see that breathing high barometric oxygen clearly has better prevention and curing effects on noise damage than breathing oxygen at normal pressures.

Because of the widespread clinical use of oxygen and high barometric oxygen, its poisonous side effects have been noted for a long time. However, experience has shown that if only the symptoms of adaptations are carefully noted, and the degree of pressure and length of each oxygen inhalation time are controlled, the poisonous side effects can be avoided or reduced, allowing oxygen and high barometric oxygen prevention and cure of noise damage.

(Illustrations three to five are on page 37 of inserts).

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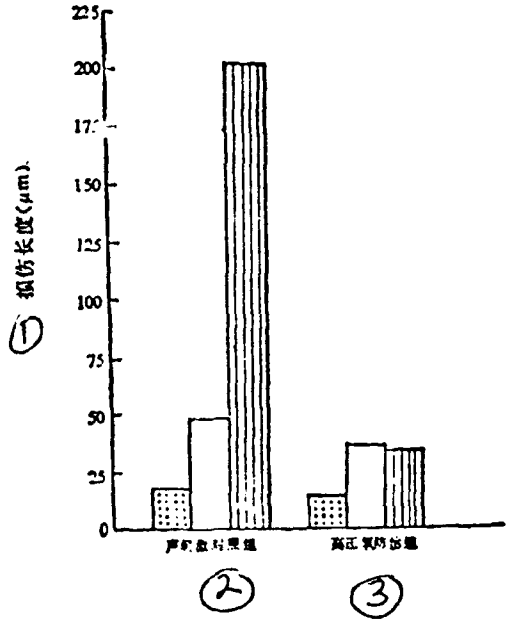

Illustration six: Length and Degree of Guinea Pig Cochlear Damage

1 - Accumulated cochlear damage;
2 - acoustic stimulation control group;
3 - high barometric oxygen group

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